09400492

* * * * * * * * * * * * * * STN Columbus * * * * * * * * * * * * * * *

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FILE 'HOME' ENTERED AT 11:21:24 ON 01 AUG 2001

=> file medline biosis embase caplus uspatfull

COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                           ENTRY    SESSION
FULL ESTIMATED COST                                         0.21       0.21

FILE 'MEDLINE' ENTERED AT 11:21:36 ON 01 AUG 2001

FILE 'BIOSIS' ENTERED AT 11:21:36 ON 01 AUG 2001
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FILE 'CAPLUS' ENTERED AT 11:21:36 ON 01 AUG 2001
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FILE 'USPATFULL' ENTERED AT 11:21:36 ON 01 AUG 2001
CA INDEXING COPYRIGHT (C) 2001 AMERICAN CHEMICAL SOCIETY (ACS)

=> s potassium (p) channel (p) interact? (p) protein

L1          1054 POTASSIUM (P) CHANNEL (P) INTERACT? (P) PROTEIN

=> s potassium (p) channel (p) interact? (p) protein (p) congestive

   4 FILES SEARCHED...
L2             0 POTASSIUM (P) CHANNEL (P) INTERACT? (P) PROTEIN (P) CONGESTIVE

=> s potassium (p) channel (p) interact? (p) protein (p) long

   4 FILES SEARCHED...
L3            50 POTASSIUM (P) CHANNEL (P) INTERACT? (P) PROTEIN (P) LONG

=> s potassium (p) channel (p) interact? (p) protein (p) long (p) qt

L4            20 POTASSIUM (P) CHANNEL (P) INTERACT? (P) PROTEIN (P) LONG (P) QT

=> dup rem l4

PROCESSING COMPLETED FOR L4
L5             8 DUP REM L4 (12 DUPLICATES REMOVED)

=> s potassium (a) channel (a) interact? (a) protein

L6            15 POTASSIUM (A) CHANNEL (A) INTERACT? (A) PROTEIN

=> dup rem l6

PROCESSING COMPLETED FOR L6
L7            12 DUP REM L6 (3 DUPLICATES REMOVED)
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=> d his

     (FILE 'HOME' ENTERED AT 11:21:24 ON 01 AUG 2001)

     FILE 'MEDLINE, BIOSIS, EMBASE, CAPLUS, USPATFULL' ENTERED AT 11:21:36 ON
     01 AUG 2001
L1          1054 S POTASSIUM (P) CHANNEL (P) INTERACT? (P) PROTEIN
L2             0 S POTASSIUM (P) CHANNEL (P) INTERACT? (P) PROTEIN (P)
CONGESTIV
L3            50 S POTASSIUM (P) CHANNEL (P) INTERACT? (P) PROTEIN (P) LONG
L4            20 S POTASSIUM (P) CHANNEL (P) INTERACT? (P) PROTEIN (P) LONG
(P)
L5             8 DUP REM L4 (12 DUPLICATES REMOVED)
L6            15 S POTASSIUM (A) CHANNEL (A) INTERACT? (A) PROTEIN
L7            12 DUP REM L6 (3 DUPLICATES REMOVED)

=> d l5 total ibib kwic


L5   ANSWER 1 OF 8     MEDLINE                              DUPLICATE 1
ACCESSION NUMBER:   1998215377     MEDLINE
DOCUMENT NUMBER:    98215377   PubMed ID: 9556090
TITLE:              Genetics, molecular mechanisms and management of long QT
                    syndrome.
AUTHOR:             Wang Q; Chen Q; Towbin J A
CORPORATE SOURCE:   Department of Pediatrics, Baylor College of Medicine,
Texas
                    Children's Hospital, Houston 77030, USA..
qwang@bcm.tmc.edu
SOURCE:             ANNALS OF MEDICINE, (1998 Feb) 30 (1) 58-65.  Ref: 63
                    Journal code: AMD; 8906388. ISSN: 0785-3890.
PUB. COUNTRY:       ENGLAND: United Kingdom
                    Journal; Article; (JOURNAL ARTICLE)
                    General Review; (REVIEW)
                    (REVIEW, TUTORIAL)
LANGUAGE:           English
FILE SEGMENT:       Priority Journals
ENTRY MONTH:        199806
ENTRY DATE:         Entered STN: 19980618
                    Last Updated on STN: 19980618
                    Entered Medline: 19980611
AB   Cardiac arrhythmias cause more than 300,000 sudden deaths each year in
the
     USA alone. Long QT syndrome (LQT) is a cardiac
     disorder that causes sudden death from ventricular tachyarrhythmias,
     specifically torsade de pointes. Four LQT genes.  .  .  (LQT2) on
     chromosome 7q35-36, SCN5A (LQT3) on chromosome 3p21-24, and MinK (LQT5)
on
     chromosome 21q22. SCN5A encodes the cardiac sodium channel, and
     LQT-causing mutations in SCN5A lead to the generation of a late phase of
     inactivation-resistant whole-cell inward currents. Mexiletine, a sodium
     channel blocker, is effective in shortening the QT
     interval corrected for heart rate (QTc) of patients with SCN5A mutations.
     HERG encodes the cardiac I(Kr) potassium channel.
     Mutations in HERG act by a dominant-negative mechanism or by a
     loss-of-function mechanism. Raising the serum potassium
     concentration can increase outward HERG potassium current and is
     effective in shortening the QTc of patients with HERG mutations. KVLQT1
is
     a cardiac potassium channel protein that
     interacts with another small potassium channel
     MinK to form the cardiac I(Ks) potassium channel. Like
     HERG mutations, mutations in KVLQT1 and MinK can act by a
     dominant-negative mechanism or a loss-of-function mechanism. An
effective.
```

. . LQT patients with KVLQT1 or MinK mutations is expected to be developed based on the functional characterization of the I(Ks) **potassium channel**. Genetic testing is now available for some patients with LQT.

L5 ANSWER 2 OF 8 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1997:506691 CAPLUS
DOCUMENT NUMBER: 127:118273
TITLE: KVLQT1 is a long QT syndrome gene whose protein product coassembles with minK to form cardiac IKs potassium channels
INVENTOR(S): Keating, Mark F.; Curran, Mark E.; Landes, Gregory M.;
Connors, Timothy D.
PATENT ASSIGNEE(S): University of Utah Research Foundation, USA; Genzyme Genetics
SOURCE: PCT Int. Appl., 116 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9723632	A1	19970703	WO 1996-US19917	19961220
W: AU, CA, JP, KR, NZ				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
AU 9719512	A1	19970717	AU 1997-19512	19961220
AU 714041	B2	19991216		
EP 87649	A1	19981111	EP 1996-946233	19961220
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 2001501445	T2	20010206	JP 1997-523726	19961220

PRIORITY APPLN. INFO.:
US 1995-19014 P 19951222
US 1996-739383 A 19961029
WO 1996-US19917 W 19961220

AB One aspect of the invention relates to the identification of the mol. basis of **long QT** syndrome. KVLQT1 cDNA was isolated from a human heart cDNA library, and its 581-amino-acid **protein** product was structurally and functionally characterized; a Xenopus KVLQT1 homolog was also identified. More specifically, mutated KVLQT1 is shown to cause **long QT** syndrome. Anal. of this gene will provide an early diagnosis of subjects with **long QT** syndrome. The diagnostic methods comprise analyzing the nucleic acid sequence of the KVLQT1 gene of an individual to be tested and comparing them with the nucleic acid sequence of the native, non-variant gene. Alternatively, the amino acid sequence of KVLQT1 may be analyzed for mutations which cause **long QT** syndrome. Presymptomatic diagnosis of **long QT** syndrome will enable practitioners to treat this disorder using existing medical therapy. A second aspect of the invention relates to the realization that KVLQT1 coassemble with minK to form a cardiac **potassium channel**. This allows one to assay for drugs which **interact** with this **channel** to identify new drug which are useful for treating or preventing **long QT**.

L5 ANSWER 3 OF 8 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1997:506918 CAPLUS
DOCUMENT NUMBER: 127:118275
TITLE: KVLQT1 is a long QT syndrome gene whose protein product coassembles with mink to form cardiac IKs potassium channels
INVENTOR(S): Keating, Mark T.; Sanguinetti, Michael C.; Curran,

Mark E.
PATENT ASSIGNEE(S): University of Utah Research Foundation, USA
SOURCE: PCT Int. Appl., 105 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9723598	A2	19970703	WO 1996-US19756	19961220
W: AU, CA, JP, KR, NZ				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
AU 9717428	A1	19970717	AU 1997-17428	19961220
AU 714527	B2	20000106		
EP 870041	A2	19981014	EP 1996-945935	19961220
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

PRIORITY APPLN. INFO.: US 1995-19014 P 19951222
US 1996-739383 A 19961029
WO 1996-US19756 W 19961220

AB One aspect of the invention relates to the identification of the mol. basis of **long QT** syndrome. KVLQT1 cDNA was isolated from a human heart cDNA library, and its 581-amino-acid **protein** product was structurally and functionally characterized; a Xenopus KVLQT1 homolog was also identified. Mutations in KVLQT1 are shown to cosegregate with the **long QT** syndrome. More specifically, minK is found to coassemble with KVQLT1 to form a cardiac **potassium channel**. Thus, mutated minK causes **long QT** syndrome. Anal. of this gene will provide an early diagnosis of subjects with **long QT** syndrome. The diagnostic methods comprise analyzing the nucleic acid sequence of the minK gene of an individual to be tested and comparing them with the nucleic acid sequence of the native, non-variant gene. Alternatively, the amino acid sequence of minK may be analyzed for mutations which cause **long QT** syndrome. Presymptomatic diagnosis of **long QT** syndrome will enable practitioners to treat this disorder using existing medical therapy. Assays are also provided for drugs which **interact** with the cardiac **potassium channel** to identify new drugs which are useful for treating or preventing **long QT**.

L5 ANSWER 4 OF 8 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 97472471 MEDLINE
DOCUMENT NUMBER: 97472471 PubMed ID: 9328483
TITLE: IsK and KvLQT1: mutation in either of the two subunits of the slow component of the delayed rectifier potassium channel can cause Jervell and Lange-Nielsen syndrome.
AUTHOR: Tyson J; Tranebjaerg L; Bellman S; Wren C; Taylor J F; Bathen J; Aslaksen B; Sorland S J; Lund O; Malcolm S; Pembrey M; Bhattacharya S; Bitner-Glindzicz M
CORPORATE SOURCE: Unit of Clinical Genetics, Institute of Child Health and Great Ormond Street Hospital for Children NHS Trust, UCL Medical School, 30 Guilford Street, London WC1N 1EH, UK.
SOURCE: HUMAN MOLECULAR GENETICS, (1997 Nov) 6 (12) 2179-85.
Journal code: BRC; 9208958. ISSN: 0964-6906.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199712
ENTRY DATE: Entered STN: 19980109
Last Updated on STN: 19990129

Entered Medline: 19971219

AB . . . sensorineural deafness associated with syncopal episodes. These are caused by ventricular arrhythmias secondary to abnormal repolarisation, manifested by a prolonged **QT** interval on the electrocardiogram. Recently, in families with JLNS, Neyroud et al. reported homozygosity for a single mutation in KVLQT1 , a gene which has previously been shown to be mutated in families with dominantly inherited isolated **long QT** syndrome [Neyroud et al . (1997) Nature Genet ., 15, 186-189]. We have analysed a group of families with JLNS. . . by descent for markers on chromosome 21, in a region containing the gene IsK . This codes for a transmembrane **protein** known to associate with KVLQT1 to form the slow component of the delayed rectifier **potassium channel**. Sequencing of the affected boys showed a homozygous mutation, demonstrating that mutation in the IsK gene may be a rare cause of JLNS and that an indistinguishable phenotype can arise from mutations in either of the two **interacting** molecules.

L5 ANSWER 5 OF 8 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 97462827 MEDLINE
DOCUMENT NUMBER: 97462827 PubMed ID: 9323054
TITLE: Dominant-negative KvLQT1 mutations underlie the LQT1 form of long QT syndrome.
COMMENT: Comment in: Circulation. 1997 Sep 16;96(6):1720-1
AUTHOR: Shalaby F Y; Levesque P C; Yang W P; Little W A; Conder M L; Jenkins-West T; Blanar M A
CORPORATE SOURCE: Department of Cardiovascular Drug Discovery, Bristol-Myers Squibb Pharmaceutical Research Institute, Princeton, NJ 08543-4000, USA.
SOURCE: CIRCULATION, (1997 Sep 16) 96 (6) 1733-6.
Journal code: DAW; 0147763. ISSN: 0009-7322.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199710
ENTRY DATE: Entered STN: 19971105
Last Updated on STN: 19990129
Entered Medline: 19971022

AB BACKGROUND: Mutations that map to the KvLQT1 gene on human chromosome 11 account for more than 50% of inherited **long QT** syndrome (LQTS). It has been discovered recently that the KvLQT1 and minK **proteins** functionally **interact** to generate a current with biophysical properties similar to I(Ks), the slowly activating delayed-rectifier cardiac **potassium** current. Since I(Ks) modulates the repolarization of cardiac action potentials it is reasonable to hypothesize that mutations in KvLQT1 reduce. . . S2-S3 cytoplasmic loop (A177P) or threonine with isoleucine in the highly conserved signature sequence of the pore (T311I) yield inactive **channels** when expressed individually, whereas substitution of leucine with phenylalanine in the S5 transmembrane domain (L272F) yields a functional **channel** with reduced macroscopic conductance. However, all these mutants inhibit wild-type KvLQT1 currents in a dominant-negative fashion. CONCLUSIONS: In LQTS-affected individuals. . .

L5 ANSWER 6 OF 8 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 97150816 MEDLINE
DOCUMENT NUMBER: 97150816 PubMed ID: 8995352
TITLE: The human delta1261 mutation of the HERG potassium channel results in a truncated protein that contains a subunit interaction domain and decreases the channel expression.
AUTHOR: Li X; Xu J; Li M
CORPORATE SOURCE: Department of Physiology, The Johns Hopkins University School of Medicine, Baltimore, Maryland 21205, USA.

SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1997 Jan 10) 272 (2) 705-8.
Journal code: HIV; 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199702
ENTRY DATE: Entered STN: 19970227
Last Updated on STN: 19970227
Entered Medline: 19970212

AB HERG (human eag-related gene) encodes an inward-rectifier **potassium channel** formed by the assembly of four subunits. Since the truncated HERG **protein** in patients with **long QT** syndrome induces a dominant phenotype, that is, cardiac sudden death, the assembly of nonfunctional complexes between wild-type and mutated subunits. . . To understand HERG-mediated cardiac sudden death at the molecular level, it is important to determine which regions in the HERG **protein** participate in subunit **interaction**. We therefore report the identification of a subunit **interaction** domain, NAB(HERG), that is localized at the hydrophilic cytoplasmic N terminus and can form a tetramer in the absence of the rest of the HERG **protein**. Truncated HERG **proteins** containing NAB(HERG), including one that resulted from the delta1261 human mutation, inhibit the functional expression of the HERG **channel** in transfected cells. Together, these results support the notion that the expression of HERG in the human heart may be decreased in the presence of the truncated subunit. Such a decrease of **potassium channel** expression can contribute to the longer **QT** intervals observed in the patients with the HERG mutation.

L5 ANSWER 7 OF 8 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 1997:175428 BIOSIS
DOCUMENT NUMBER: PREV199799467141
TITLE: Building (potassium) channels to the 21st century (Neuropharmacology IV: K+ channels: Washington, D.C., USA, November 14-16, 1996.
AUTHOR(S): Trimmer, James S. (1); Rhodes, Kenneth J.
CORPORATE SOURCE: (1) Dep. Biochemistry Cell Biology, Inst. Cell Dev. Biology, State Univ. New York Stony Brook, Stony Brook, NY 11794 USA
SOURCE: Trends in Neurosciences, (1997) Vol. 20, No. 3, pp. 99-100.
ISSN: 0166-2236.
DOCUMENT TYPE: Conference; Report
LANGUAGE: English
IT Miscellaneous Descriptors
BIOCHEMISTRY AND BIOPHYSICS; GENOME SEQUENCE; HEART DISEASE; **LONG QT** SYNDROME; **POTASSIUM CHANNELS; PROTEIN-PROTEIN INTERACTIONS;** RESTING MEMBRANE POTENTIAL; SHAKER **PROTEIN**

L5 ANSWER 8 OF 8 MEDLINE
ACCESSION NUMBER: 95406767 MEDLINE
DOCUMENT NUMBER: 95406767 PubMed ID: 7676324
TITLE: Molecular genetics of long QT syndrome.
AUTHOR: Keating M T
CORPORATE SOURCE: Department of Human Genetics, University of Utah, Salt Lake City 84112, USA.
CONTRACT NUMBER: RO1HL48074 (NHLBI)
SOURCE: SOCIETY OF GENERAL PHYSIOLOGISTS SERIES, (1995) 50 53-60.
Ref: 28

Journal code: UU2; 0433431. ISSN: 0094-7733.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199510
ENTRY DATE: Entered STN: 19951026
Last Updated on STN: 19951026
Entered Medline: 19951019

AB In the **long QT** syndrome (LQT), individuals suffer from syncope, seizures and sudden death due to cardiac arrhythmias, specifically torsade de pointes and ventricular fibrillation. Many of these individuals also have prolongation of the **QT** interval on electrocardiograms, suggesting abnormal cardiac repolarization. To improve our understanding of the mechanisms underlying LQT and to facilitate presymptomatic. . . making this protoncogene a candidate for LQT. This hypothesis was supported by physiologic data; other investigators had shown that ras **proteins** modulate cardiac **potassium channels** and an abnormality of **potassium** homeostasis could explain LQT. We eliminated HRAS as a candidate, however, by sequencing the coding region in 10 unrelated patients. . . unlinked, at least one more LQT locus exists. This degree of heterogeneity presents opportunities. It seems likely, for example, that **proteins** encoded by distinct LQT genes **interact** to modulate cardiac repolarization.(ABSTRACT TRUNCATED AT 250 WORDS)

=> d l7 total ibib kwic

L7 ANSWER 1 OF 12 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2001336578 MEDLINE
DOCUMENT NUMBER: 21297487 PubMed ID: 11404400
TITLE: Kinetic modulation of Kv4-mediated A-current by arachidonic acid is dependent on **potassium channel interacting proteins**.
AUTHOR: Holmqvist M H; Cao J; Knoppers M H; Jurman M E; Distefano P S; Rhodes K J; Xie Y; An W F
CORPORATE SOURCE: Millennium Pharmaceuticals Inc., Cambridge, Massachusetts 02139, USA.
SOURCE: JOURNAL OF NEUROSCIENCE, (2001 Jun 15) 21 (12) 4154-61.
Journal code: JDF; 8102140. ISSN: 1529-2401.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200107
ENTRY DATE: Entered STN: 20010716
Last Updated on STN: 20010716
Entered Medline: 20010712

TI Kinetic modulation of Kv4-mediated A-current by arachidonic acid is dependent on **potassium channel interacting proteins**.

L7 ANSWER 2 OF 12 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 2001:199211 BIOSIS
DOCUMENT NUMBER: PREV200100199211
TITLE: K-Channel Interacting Protein dependent modulation of Kv4 current by fatty acids.
AUTHOR(S): Holmqvist, Mats H. (1); Cao, Jie (1); Jurman, Mark E. (1);

Xie, Yu (1); Rhodes, Kenneth J.; Distefano, Peter S. (1); An, W. Frank (1)
CORPORATE SOURCE: (1) Millennium Pharmaceuticals, Inc., Cambridge, MA, 02139 USA
SOURCE: Biophysical Journal, (January, 2001) Vol. 80, No. 1 Part 2,
pp. 506a. print.
Meeting Info.: 45th Annual Meeting of the Biophysical Society Boston, Massachusetts, USA February 17-21, 2001 Biophysical Society
. ISSN: 0006-3495.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English
IT . . .
neuron: dendritic A current, nervous system
IT Chemicals & Biochemicals
Kv4 alpha-subunit; Kv4 potassium channel: modulation; arachidonic acid;
fatty acids; **potassium channel-interacting protein**

L7 ANSWER 3 OF 12 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 2001:173636 BIOSIS
DOCUMENT NUMBER: PREV200100173636
TITLE: Kv4 channel gating and effects of the K channel interacting
proteins (KChIPs.
AUTHOR(S): Baehring, Robert (1); Gebauer, Manuel (1); Boland, Linda M.
(1); Pongs, Olaf (1); Isbrandt, Dirk (1)
CORPORATE SOURCE: (1) Institut fuer Neurale Signalverarbeitung, Martinistr. 52, 20246, Hamburg Germany
SOURCE: Biophysical Journal, (January, 2001) Vol. 80, No. 1 Part 2,
pp. 439a. print.
Meeting Info.: 45th Annual Meeting of the Biophysical Society Boston, Massachusetts, USA February 17-21, 2001 Biophysical Society
. ISSN: 0006-3495.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English
IT Major Concepts
Biochemistry and Molecular Biophysics; Cell Biology
IT Chemicals & Biochemicals
Kv4 channel: gating; **potassium channel interaction protein** [KCHIPs]

L7 ANSWER 4 OF 12 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2001219091 EMBASE
TITLE: Different effects of the Ca(2+)-binding protein, KChIP1, on
two Kv4 subfamily members, Kv4.1 and Kv4.2.
AUTHOR: Nakamura T.Y.; Nandi S.; Pountney D.J.; Artman M.; Rudy B.;
Coetzee W.A.
CORPORATE SOURCE: T.Y. Nakamura, Pediatric Cardiology (TH519), NYU School of Medicine, 560 First Avenue, New York, NY 10016, United States. tomoe.nishitani@med.nyu.edu
SOURCE: FEBS Letters, (22 Jun 2001) 499/3 (205-209).
Refs: 27
ISSN: 0014-5793 CODEN: FEBLAL
PUBLISHER IDENT.: S 0014-5793(01)02560-1
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
CT Medical Descriptors:
*protein . . . interaction
protein family
potassium channel
potassium current
chimera
amino terminal sequence
biodiversity
protein expression
nonhuman
controlled study
animal cell
article
nucleotide sequence
priority journal
*calcium binding protein: EC, endogenous compound
*protein kv4: EC, endogenous compound
***potassium channel interacting protein 1: EC, endogenous compound**
*protein subunit: EC, endogenous compound
unclassified drug

L7 ANSWER 5 OF 12 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 2001:208654 BIOSIS
DOCUMENT NUMBER: PREV200100208654
TITLE: Molecular cloning and expression of the novel splice variants of K+ channel-interacting protein 2.
AUTHOR(S): Ohya, Susumu; Morohashi, Yuichi; Muraki, Katsuhiko; Tomita, Taisuke; Watanabe, Minoru; Iwatsubo, Takeshi; Imaizumi, Yuji (1)
CORPORATE SOURCE: (1) Department of Molecular and Cellular Pharmacology, Faculty of Pharmaceutical Sciences, Nagoya City University, 3-1 Tanabedori, Mizuhoku, Nagoya, 467-8603: yimaizum@phar.nagoya-cu.ac.jp Japan
SOURCE: Biochemical and Biophysical Research Communications, (March 23, 2001) Vol. 282, No. 1, pp. 96-102. print. ISSN: 0006-291X.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English
IT . . .
Organisms
aorta: circulatory system; brain: nervous system; heart: circulatory system; kidney: excretory system
IT Chemicals & Biochemicals
cDNA [complementary DNA]; **potassium channel-interacting protein** 2 [KChIP2]: expression, molecular cloning

L7 ANSWER 6 OF 12 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 2000:368431 CAPLUS
DOCUMENT NUMBER: 133:13914
TITLE: Proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining
INVENTOR(S): Rhodes, Kenneth; Betty, Maria; Ling, Huai-ping; An, Wenqian
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc., USA; American Home Products Corporation
SOURCE: PCT Int. Appl., 306 pp. CODEN: PIXXD2
DOCUMENT TYPE: Patent

LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000031133	A2	20000602	WO 1999-US27428	19991119
WO 2000031133	A3	20001005		

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ

RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.:

US 1998-109333	P	19981120
US 1998-110033	P	19981125
US 1998-110277	P	19981130
US 1999-298731	A2	19990423
US 1999-350614	A2	19990709
US 1999-350874	A2	19990709
US 1999-399913	A2	19990921
US 1999-400492	A2	19990921

ST **potassium channel interacting protein** gene cloning; mouse monkey **potassium channel interacting protein**; human rat **potassium channel interacting protein**; calcium binding **potassium channel interacting protein**

IT Proteins, specific or class
RL: BOC (Biological occurrence); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(PCIP (**potassium-channel interacting proteins**); proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT Nervous system
(central, disease, effectors of **potassium channel-interacting protein** for treatment of; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT Brain
(cerebellum, granular layer, **potassium-channel interacting proteins** mRNA in; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT Brain
(corpus striatum, **potassium-channel interacting proteins** mRNA in; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT Brain
(cortex, **potassium-channel interacting proteins** mRNA in; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT Cardiovascular system
(disease, effectors of **potassium channel-interacting protein** for treatment of; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT Anticonvulsants
(effectors of **potassium channel-interacting protein** as; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT Epilepsy
(effectors of **potassium channel-interacting**

protein for treatment of; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT Nucleic acid amplification (method)
Nucleic acid hybridization
(for detection of **potassium channel-interacting protein** gene expression; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT Primers (nucleic acid)
Probes (nucleic acid)
RL: ARG (Analytical reagent use); ANST (Analytical study); USES (Uses)
(for detection of **potassium channel-interacting protein** gene expression; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT Immunoassay
(for detection of **potassium channel-interacting proteins**; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT Susceptibility (genetic)
(for disease assocd. with **potassium channel-interacting protein**, screening for; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT Drug screening
(for effectors of **potassium channel-interacting protein**; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT Gene, animal
RL: BOC (Biological occurrence); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(for **potassium channel interacting proteins**; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT cDNA sequences
(for **potassium channel-interacting proteins** of human, mouse, rat, and monkey; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT mRNA
RL: BOC (Biological occurrence); BIOL (Biological study); OCCU (Occurrence)
(for **potassium-channel interacting proteins**, tissue distribution of; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT Brain
(habenula, medial nucleus, **potassium-channel interacting proteins** mRNA in; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT Brain
(hippocampus, **potassium-channel interacting proteins** mRNA in; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT Chromosome
(human 10, 10q22-q24, **potassium-channel interacting protein** gene mapping to; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT EF hand
(in **potassium-channel interacting proteins**; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT Brain
(midbrain, **potassium-channel interacting proteins** mRNA in; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)
IT Diagnosis
(mol., of disease assocd. with **potassium channel-interacting protein**; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)
IT Molecular cloning
(of cDNAs for **potassium channel-interacting proteins** of human, mouse, rat, and monkey; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)
IT Protein sequences
(of **potassium channel-interacting proteins** of human, mouse, rat, and monkey; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)
IT Genetic mapping
(of **potassium-channel interacting protein** gene of human; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)
IT Calcium-binding **proteins**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**potassium-channel interacting proteins** as; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)
IT Brain
Heart
(**potassium-channel interacting proteins** mRNA in; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)
IT Nervous system
(spinocerebellar ataxia, effectors of **potassium channel-interacting protein** for treatment of; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)
IT Brain
(stem, **potassium-channel interacting proteins** mRNA in; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)
IT Brain
(substantia nigra, **potassium-channel interacting proteins** mRNA in; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)
IT Brain
(superior colliculus, **potassium-channel interacting proteins** mRNA in; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)
IT Brain
(thalamus, **potassium-channel interacting proteins** mRNA in; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)
IT Antibodies
RL: ARG (Analytical reagent use); ANST (Analytical study); USES (Uses)
(to **potassium channel-interacting proteins**; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

L7 ANSWER 7 OF 12 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 2001:101622 BIOSIS
DOCUMENT NUMBER: PREV200100101622
TITLE: Kinetic modulation of Kv4-current by arachidonic acid is

dependent on K-channel interacting proteins.
AUTHOR(S): Holmqvist, M. H. (1); Xie, Y.; Jurman, M. E.; Cao, J.; Rhodes, K. J.; Distefano, P. S.; An, W. F.
CORPORATE SOURCE: (1) Millennium Pharmaceuticals, Inc., Cambridge, MA USA
SOURCE: Society for Neuroscience Abstracts, (2000) Vol. 26, No. 1-2, pp. Abstract No.-614.3. print.
Meeting Info.: 30th Annual Meeting of the Society of Neuroscience New Orleans, LA, USA November 04-09, 2000 Society for Neuroscience
. ISSN: 0190-5295.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English
IT . . .
Biochemistry and Molecular Biophysics
IT Parts, Structures, & Systems of Organisms
oocytes: reproductive system
IT Chemicals & Biochemicals
arachidonic acid; **potassium-channel interacting proteins**; voltage-gated fast-activating potassium channels: inactivation

L7 ANSWER 8 OF 12 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 2001:102702 BIOSIS
DOCUMENT NUMBER: PREV200100102702
TITLE: K-channel interacting protein-2 splice variants, chromosomal organization and localization.
AUTHOR(S): An, W. F. (1); Ling, H. P.; Chen, H.; Xie, Y.; Holmqvist, M. H.; Cao, J.; Jurman, M. E.; Dussault, B. J.; Distefano, P. S.; Betty, M.; Mendoza, G.; Bowlby, M. R.; Rhodes, K. J.
CORPORATE SOURCE: (1) Millennium Pharmaceuticals, Cambridge, MA USA
SOURCE: Society for Neuroscience Abstracts, (2000) Vol. 26, No. 1-2, pp. Abstract No.-614.2. print.
Meeting Info.: 30th Annual Meeting of the Society of Neuroscience New Orleans, LA, USA November 04-09, 2000 Society for Neuroscience
. ISSN: 0190-5295.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English
IT Major Concepts
Molecular Genetics (Biochemistry and Molecular Biophysics)
IT Chemicals & Biochemicals
cDNA [complementary DNA]; **potassium-channel interacting protein**-2: chromosomal organization, localization, splice variants; voltage-gated fast-inactivating potassium channels

L7 ANSWER 9 OF 12 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 2000:27272 CAPLUS
DOCUMENT NUMBER: 132:149426
TITLE: Caspr2, a new member of the neurexin superfamily, is localized at the juxtaparanodes of myelinated axons and associates with K+ channels
AUTHOR(S): Poliak, Sebastian; Gollan, Leora; Martinez, Ricardo; Custer, Andrew; Einheber, Steven; Salzer, James L.; Trimmer, James S.; Shrager, Peter; Peles, Elior
CORPORATE SOURCE: Department of Molecular Cell Biology, The Weizmann Institute of Science, Rehovot, 76100, Israel
SOURCE: Neuron (1999), 24(4), 1037-1047
CODEN: NERNET; ISSN: 0896-6273
PUBLISHER: Cell Press
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 53

REFERENCE(S): (1) Baumgartner, S; Cell 1996, V87, P1059 CAPLUS
(2) Bekele-Arcuri, Z; Neuropharmacology 1996, V35, P851 CAPLUS
(3) Bellen, H; Trends Neurosci 1998, V21, P444 CAPLUS
(5) Bhat, M; Cell 1999, V96, P833 CAPLUS
(6) Butz, S; Cell 1998, V94, P773 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

ST protein Caspr2 juxtaparanode myelinated axon; **potassium channel interaction protein** Caspr2 myelinated axon; human cDNA sequence caspr2 protein

L7 ANSWER 10 OF 12 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 1999:186364 BIOSIS
DOCUMENT NUMBER: PREV199900186364
TITLE: Molecular identification and functional characterization of
KIP1 and KIP2.
AUTHOR(S): Gong, Jianping (1); Xu, Jia (1); Bezanilla, Magdalena (1); Derin, Rachel (1); Li, Min (1)
CORPORATE SOURCE: (1) Department of Physiology and Department of Neuroscience, The Johns Hopkins University School of Medicine, 725 North Wolfe Street, Baltimore, MD, 21205 USA
SOURCE: Biophysical Journal, (Jan., 1999) Vol. 76, No. 1 PART 2, pp. A346.
Meeting Info.: Forty-third Annual Meeting of the Biophysical Society Baltimore, Maryland, USA February 13-17, 1999
ISSN: 0006-3495.
DOCUMENT TYPE: Conference
LANGUAGE: English
IT Major Concepts
Biochemistry and Molecular Biophysics
IT Chemicals & Biochemicals
potassium channel; KIP1: **potassium channel interacting protein**; KIP2: **potassium channel interacting protein**

L7 ANSWER 11 OF 12 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1997:259948 CAPLUS
DOCUMENT NUMBER: 126:340102
TITLE: Cytoplasmic domains of voltage-sensitive K+ channels involved in mediating protein-protein interactions
AUTHOR(S): Scannevin, Robert H.; Trimmer, James S.
CORPORATE SOURCE: Cell Biology Inst. Cell Developmental Biology, State Univ. New York, Stony Brook, NY, 11794-5215, USA
SOURCE: Biochem. Biophys. Res. Commun. (1997), 232(3), 585-589
CODEN: BBRCA9; ISSN: 0006-291X
PUBLISHER: Academic
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
ST review voltage sensitive potassium channel domain; **protein interaction potassium channel** domain review

L7 ANSWER 12 OF 12 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1995:79211 CAPLUS
DOCUMENT NUMBER: 122:1513
TITLE: The molecular regulation of muscarinic K+ channels by GTP-binding proteins in cardiac atrial cell membrane
AUTHOR(S): Kurachi, Yoshihisa; Ito, Hiroyuki; Takikawa, Reiko; Nakajima, Toshiaki; Sugimoto, Tsuneaki
CORPORATE SOURCE: Dep. Int. Med., Mayo Clinic, Rochester, MN, 55905, USA
SOURCE: Mol. Biol. Myocard. (1992), 189-202. Editor(s): Tada,
Michihiko. Jpn. Sci. Soc. Press: Tokyo, Japan.

CODEN: 60MQAG
DOCUMENT TYPE: Conference
LANGUAGE: English
IT G proteins (guanine nucleotide-binding **proteins**)
RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)
(**potassium channels interaction** with adenosine receptors regulation by G proteins and their subunits in cardiac atrial cell membrane)

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